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=> s erythrocyte adenylate kinase/cn
L1            0 ERYTHROCYTE ADENYLATE KINASE/CN

=> s adenylate kinase/cn
L2            1 ADENYLATE KINASE/CN

=> file ca
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                                                       ENTRY    SESSION
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(=>) for specific information.

=> s k2
L3       15217 K2

=> s l2
L4        1879 L2

=> s l4 and (hemolysis)/ab,bi
          7731 (HEMOLYSIS)/AB
         10406 (HEMOLYSIS)/BI
L5          17 L4 AND (HEMOLYSIS)/AB,BI

=> s l5 and (erythrocyt? or red(2w)cell#)/ab,bi
         65607 ERYTHROCYT?/AB
         83648 ERYTHROCYT?/BI
        136311 RED/AB
        152718 RED/BI
       1162994 CELL#/AB
       1308055 CELL#/BI
         30230 RED(2W)CELL#
L6          13 L5 AND (ERYTHROCYT? OR RED(2W)CELL#)/AB,BI

=> d bib,ab 1-13

L6   ANSWER 1 OF 13  CA  COPYRIGHT 1997 ACS
AN   122:26347  CA
TI   Adenylate kinase mimics creatine kinase-MM isoenzyme in a CK
     isoenzyme electrophoresis assay
AU   Murthy, Vadiraja V.
CS   Dep. Lab. Med., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SO   J. Clin. Lab. Anal. (1994), 8(3), 140-3
     CODEN: JCANEM; ISSN: 0887-8013
DT   Journal
LA   English
AB   Adenylate kinase (AK) activity originating from erythrocytes
     , present in hemolyzed serum, behaves like creatine kinase MM
     isoenzyme (CK-MM) in some CK electrophoresis assays that employ, in
     their visualization reagent kits, AMP as the sole inhibitor of AK,
     rather than a combination of AMP and a more potent inhibitor of
     erythrocyte AK, diadenosine pentaphosphate (Ap5A), to
     inhibit all contaminating AK activities in serum and quantify only
     the CK isoenzyme activities in serum following electrophoretic
     fractionation on agarose gel.  This can spuriously overestimate the
     CK-MM fraction and thereby result in underestimation of CK-MB or
     CK-BB isoenzymes if present.  A hemolyzed serum sample obtained from
     an elderly patient was erroneously reported as contg. low CK-MB due
     to such overestimation of CK-MM fraction in the sample.
```

Supplementing the AMP already present in the visualization reagent formulation, used to est. CK isoenzyme concn. in serum, with Ap5A can eliminate or effectively minimize AK interference, esp. that caused by **hemolysis**, and thereby prevent reporting false-neg. CK-MB results obtained with CK isoenzyme electrophoresis assays.

L6 ANSWER 2 OF 13 CA COPYRIGHT 1997 ACS
AN 117:168738 CA
TI Decreased pyrimidine nucleoside monophosphate kinase activity in sickle **erythrocytes**
AU Zerez, Charles R.; Lachant, Neil A.; Lent, Kathleen M.; Tanaka, Kouichi R.
CS Med. Cent., Harbor-UCLA, Torrance, CA, USA
SO Blood (1992), 80(2), 512-16
CODEN: BLOOAW; ISSN: 0006-4971
DT Journal
LA English
AB The authors have previously shown that physiol. concns. of hemin cause marked inhibition of several **red** blood **cell** (RBC) enzymes. Because endogenous heme content is elevated in sickle RBCs, the authors examd. the activity of hemin-sensitive enzymes in these RBCs. One of the hemin-sensitive enzymes, pyrimidine nucleoside monophosphate kinase (PNMK), was shown to have decreased activity in sickle RBCs relative to RBCs of equiv. cell age. The other hemin-sensitive enzymes, including adenylate kinase (AK), pyrimidine 5'-nucleotidase (P5N), 6-phosphogluconate dehydrogenase (6PGD), and aldolase, had activities that were appropriate for cell age. The affinity of the hemin-sensitive enzymes to hemin was also examd. Using two different methods, PNMK was shown to have the highest binding affinity to hemin. The exquisite sensitivity of PNMK to inhibition by hemin, coupled with the enzyme's high affinity to hemin, may account for the decrease in PNMK activity and the lack of significant decrease in the other hemin-sensitive enzymes in sickle RBCs. Thus, the increased endogenous heme content in sickle RBCs may be responsible for the decrease in PNMK activity. Whether the increased endogenous heme content of sickle RBCs can cause **hemolysis** indirectly by inhibiting RBC enzymes remains to be detd.

L6 ANSWER 3 OF 13 CA COPYRIGHT 1997 ACS
AN 109:36002 CA
TI Inhibition of **red** blood **cell** enzymes by hemin: a mechanism for **hemolysis** in hemoglobinopathies
AU Zerez, Charles R.; Hseih, Jasmine W.; Tanaka, Kouichi R.
CS Sch. Med., UCLA, Torrance, CA, 90509, USA
SO Trans. Assoc. Am. Physicians (1987), 100, 329-38
CODEN: TAAPAI; ISSN: 0066-9458
DT Journal
LA English
AB Hemin inhibited several enzymes of human **erythrocyte** hemolyzates. Hemin was also a potent inhibitor of the hemin-sensitive enzymes in partially purified prepns. These and other results are discussed in light of mechanisms for **hemolysis** in hemoglobinopathies.

L6 ANSWER 4 OF 13 CA COPYRIGHT 1997 ACS
AN 105:95477 CA
TI Acute intravascular **hemolysis** in the black rhinoceros: **erythrocyte** enzymes and metabolic intermediates
AU Paglia, Donald E.; Valentine, William N.; Miller, R. Eric; Nakatani, Misae; Brockway, Richard A.
CS Cent. Health Serv., Univ. California, Los Angeles, CA, 90024, USA
SO Am. J. Vet. Res. (1986), 47(6), 1321-5
CODEN: AJVRAH; ISSN: 0002-9645
DT Journal
LA English
AB Enzymes of aerobic and anaerobic glycolysis, glutathione cycling,

and nucleotide metab. were assayed on **erythrocytes** from 7 healthy rhinoceroses, 2 rhinoceroses during periods of intravascular **hemolysis**, and 1 rhinoceros without clin. signs of illness, which was the mother of 3 offspring with intravascular hemolytic syndrome. Measurements also were made of **erythrocyte** concns. of glycolytic intermediates, adenine nucleotides, and glutathione. Although comparison of results for healthy and affected rhinoceroses did not identify an enzyme abnormality as a cause for the hemolytic syndrome, the data provided information regarding the metabolic characteristics of **erythrocytes** from healthy rhinoceroses.

L6 ANSWER 5 OF 13 CA COPYRIGHT 1997 ACS
AN 99:103152 CA
TI Metabolic compensation for profound **erythrocyte** adenylate kinase deficiency. A hereditary enzyme defect without hemolytic anemia
AU Beutler, Ernest; Carson, Dennis; Dannawi, Hassan; Forman, Linda; Kuhl, Wanda; West, Carol; Westwood, Beryl
CS Dep. Basic Clin. Res., Scripps Clin. Res. Found., La Jolla, CA, 92037, USA
SO J. Clin. Invest. (1983), 72(2), 648-55
CODEN: JCINAO; ISSN: 0021-9738
DT Journal
LA English
AB A child with hemolytic anemia had severe **erythrocyte** adenylate kinase (AK) deficiency, but an equally enzyme-deficient sibling had no evidence of **hemolysis**. No residual enzyme activity was found in **erythrocytes** by spectrophotometric methods that could have detected 0.1% of normal activity. However, concd. hemolyzates had the capacity to generate small amts. of ATP and AMP from ADP. Hemolyzates could also catalyze the transfer of labeled .gamma.-phosphate from ATP to ADP. Intact **erythrocytes** were able to transfer phosphate from the .gamma.-position of ATP to the .beta.-position, albeit at a rate substantially slower than normal. They could also incorporate 14C-labeled adenine into ADP and ATP. Thus, a small amt. of residual AK-like activity representing .apprx.1/2000 of the activity normally present could be documented in the deficient **erythrocytes**. The residual activity was not inhibited by N-ethylmaleimide, which completely abolishes the activity of the normal AK1 isozyme of **erythrocytes**. The minute amt. of residual activity in **erythrocytes** could represent a small amt. of the AK2 isozyme, which as not been thought to be present in **erythrocytes**, or the activity of **erythrocyte** guanylate kinase with AMP substituting as substrate for GMP. Peripheral blood leukocytes, cultured skin fibroblasts, and transformed lymphoblasts from the deficient subject manifested .apprx.17, 24, and 74%, resp., of the activity of controls. This residual activity is consistent with the existence of genetically independent AK isozyme, AK2, which exists in these tissues. The cause of **hemolysis** in the proband may be due to an unrelated enzyme deficiency or other **erythrocyte** enzyme defect and interaction of another unidentified defect with AK deficiency.

L6 ANSWER 6 OF 13 CA COPYRIGHT 1997 ACS
AN 97:36686 CA
TI Energy metabolism in the **erythrocytes** of thoroughbred horses connected with perinatal physiological **hemolysis**
AU Medeiros, L. F.; Medeiros, L. O.; Berciano Sanjurjo, M. A.
CS Dep. Histol. Embriol., Univ. Sao Paulo, Sao Paulo, Brazil
SO Comp. Biochem. Physiol. B (1982), 71B(3), 541-4
CODEN: CBPBB8; ISSN: 0305-0491
DT Journal
LA English
AB **Erythrocyte** enzyme activities and reduced glutathione and glycolytic intermediates were detd. in newborn horses from birth to

1-mo-old and in adults. Emphasis was put on the hemolytic period at which phosphofructokinase, glutathione peroxidase, and glutathione played a significant role, glucose consumption was lower, and very high concns. of 2,3-diphosphoglycerate and ATP were detected. The metabolic adjustments may have been achieved by an increased activity of the hexose monophosphate shunt, glyceraldehyde-3-phosphate dehydrogenase, and adenylate kinase.

L6 ANSWER 7 OF 13 CA COPYRIGHT 1997 ACS
AN 93:64293 CA
TI Increased creatine kinase activities associated with **hemolysis**
AU Bais, Renze; Edwards, J. B.
CS Div. Clin. Chem., Inst. Med. Vet. Sci., Adelaide, Australia
SO Pathology (1980), 12(2), 203-7
CODEN: PTLGAX; ISSN: 0031-3025
DT Journal
LA English
AB The presence of adenylate kinase released from **erythrocytes** on **hemolysis** increases the apparent creatine kinase activity in blood serum. This can be overcome by the addn. of 10 .mu.M diadenosine pentaphosphate to the reagents used for the creatine kinase assay. The pentaphosphate strongly inhibits the **erythrocyte** adenylate kinase.

L6 ANSWER 8 OF 13 CA COPYRIGHT 1997 ACS
AN 90:135621 CA
TI The effect of external ADP on **red cell** nucleotide levels
AU Pangalis, G.; Tegos, C.; Beutler, E.
CS Sch. Med., Univ. Athens, Athens, Greece
SO Proc. Soc. Exp. Biol. Med. (1979), 160(1), 74-5
CODEN: PSEBAA; ISSN: 0037-9727
DT Journal
LA English
AB Incubation of human **erythrocytes** with radioactive and nonradioactive ADP failed to provide any evidence of the entry of this nucleotide into the cells. There was a rise in ADP levels in the cells after incubation with exogenous unlabeled ADP, but this occurred at the expense of ATP and AMP, so that the size of the total intracellular nucleotide pool diminished. The conversion of ADP into ATP and AMP in the external medium could readily be accounted for on the basis of **hemolysis** of **erythrocytes**, which contain large amts. of adenylate kinase.

L6 ANSWER 9 OF 13 CA COPYRIGHT 1997 ACS
AN 84:54900 CA
TI Effect of low-level lead exposure on pyrimidine 5'-nucleotidase and other **erythrocyte** enzymes. Possible role of pyrimidine 5'-nucleotidase in the pathogenesis of lead-induced anemia
AU Paglia, Donald E.; Valentine, William N.; Dahlgren, James G.
CS Sch. Med., Univ. California, Los Angeles, Calif., USA
SO J. Clin. Invest. (1975), 56(5), 1164-9
CODEN: JCINAO
DT Journal
LA English
AB Similarities between lead [7439-92-1]-induced anemia and a new hereditary erythroenzymopathy involving pyrimidine 5'-nucleotidase [57534-74-4] prompted studies of the effects of lead on this and other **erythrocyte** enzymes. In vitro incubations of normal mature **erythrocytes** demonstrated that significant inhibition of pyrimidine 5'-nucleotidase occurred in the presence of lead at concns. that had minimal effects on many other **erythrocyte** enzymes assayed simultaneously. Similarly, subjects with chronic lead intoxication secondary to industrial exposure exhibited substantial and consistent impairment of **erythrocyte** pyrimidine-5'-nucleotidase activity. Results suggest that lead-induced deficiency of this enzyme in maturing

erythroid elements could, if sufficiently severe, result in induction of basophilic stippling and premature **erythrocyte hemolysis** analogous to that encountered in the genetically induced enzyme-deficiency syndrome.

L6 ANSWER 10 OF 13 CA COPYRIGHT 1997 ACS
AN 84:27740 CA
TI Effects of blood sampling on enzyme activities in the serum of small laboratory animals
AU Friedel, R.; Trautschold, I.; Gaertner, K.; Helle-Feldmann, M.; Gaudssuhn, D.
CS Inst. Klin. Biochem., Med. Hochsch. Hannover, Hannover, Ger.
SO Z. Klin. Chem. Klin. Biochem. (1975), 13(11), 499-505
CODEN: ZKCKAD
DT Journal
LA German
AB Compared to the other sampling sites used, the activities of lactate dehydrogenase, malate dehydrogenase, aspartate aminotransferase, pyruvate kinase, creatine kinase, and myokinase in the blood serum of rats were high when ventral aorta and retroorbital venous plexus were used for sampling. Similar results were obtained for mouse serum enzymes after ventral aorta compared with jugular vein sampling. Alk. phosphatase and alanine aminotransferase activities in rat serum were slightly elevated after retroorbital venous plexus sampling. Small differences in plasma protein concn. and hematocrit values seen in retroorbital venous and ventral aorta sampling in rats were due to acute shifts of water within the extracellular space. Apparently, efflux of enzymes from the intracellular to the extracellular space is the main source of error (variation) in the various blood sampling techniques. The errors due to **hemolysis** and blood clotting are probably minor. The most reliable method for blood sampling of rats and mice is evidently cannulation of the jugular vein, although hert puncture may also be used.

L6 ANSWER 11 OF 13 CA COPYRIGHT 1997 ACS
AN 83:159849 CA
TI Synthetic inhibitors of adenylate kinases in the assays for ATPases and phosphokinases
AU Feldhaus, Peter; Froehlich, Thomas; Goody, Roger S.; Isakov, Maria; Schirmer, R. Heiner
CS Max-Planck-Inst. Med. Forsch., Heidelberg, Ger.
SO Eur. J. Biochem. (1975), 57(1), 197-204
CODEN: EJBCAI
DT Journal
LA English
AB Procedures are given for the synthesis of .alpha.,.omega.-dinucleoside 5'-polyphosphates as inhibitors of adenylate kinase(I). The following order for the inhibition of pig muscle I was obsd.: Ap5A >1:N6-etheno-Ap5A > Ap6A > Gp5A > Ap4A > Up5A. The synthesis of adenosine tetraphosphate, the starting material for Ap5A is also described. One mol. of pig muscle I bound 1 mol. of Ap5A. The difference spectrum of Ap5A-I with its max. of 5050 M-1 cm-1 at 271 nm, as well as the fluorescence properties of 1:N6-etheno-Ap5A can be used for kinetic binding studies. The specific binding of the neg. charged Ap5A was exploited in the prepn. of human muscle I. The enzyme was purified to homogeneity with an overall yield of 65%, the abs. value being 70 mg/kg of muscle. The effect of Ap5A on I in exts. of various cells and cell organelles was tested. A ratio of 1:50 (mole/mole) for Ap5A to other nucleotides was used for suppressing the I activity in exts. of mammalian and insect skeletal muscle, of human **erythrocytes** and of Staphylococcus aureus. A ratio of 1:5 was necessary for the I from tobacco leaves and spinach chloroplasts, and a ratio of 2:1 was needed for suppressing the I from bovine liver mitochondria, human kidney homogenate and Escherichia coli. Ap5A did not appear to be metabolized in any of the above exts. These results indicate that Ap5A can be used for evaluating the contribution of I to the prodn.

of ATP from ADP in energy-transducing systems. Contaminating I can be inhibited by a concn. of Ap5A which does not interfere in the study of many kinases and ATPases. The applications of Ap5A in the assay for nucleoside diphosphokinase and in the study of mech. and biochem. properties of contractile proteins are representative examples. The use of Ap5A makes it possible to study the effect of ADP per se in such systems. Sepharose-bound Ap5A was used for removing traces of I from samples of myosin and creatine kinase. In the presence of Ap5A, creatine kinase was measured in hemolytic serum of venous blood, in plasma of capillary blood, and in samples of whole blood after complete **hemolysis** had been induced. The clin. significance of these findings are shown for cases of myocardial infarction and muscular dystrophy.

L6 ANSWER 12 OF 13 CA COPYRIGHT 1997 ACS
AN 78:107073 CA
TI Molecular sieving of **red cell** membranes during gradual osmotic **hemolysis**
AU MacGregor, Roderick D., II; Tobias, Cornelius A.
CS Donner Lab., Univ. California, Berkeley, Calif., USA
SO J. Membrane Biol. (1972), 10(3-4), 345-56
CODEN: JMBBBO
DT Journal
LA English
AB Rat red blood corpuscles were held stationary with respect to a continuously flowing soln. in either a specially constructed centrifuge or in glass filters. The concn. of the soln. was gradually decreased to cause the swelling and subsequent gradual osmotic **hemolysis** of the cells. The passage of the intracellular mols. K, adenylate kinase, and Hb, across the cell membrane and into the flowing soln. was detd. as a function of time. Ions and mols. begin passage across the membranes in the order of increasing mol. size. The inital flow of K is followed by the initial flows of Hb and adenylate kinase. The flow of Hb was interpreted as the flows of Hb monomers, dimers and tetramers such that the time sequence is: K, Hb monomer, adenylate kinase/Hb dimer, and finally, Hb tetramer. Thus, the stressed cell membrane has mol. sieving properties and the exclusion limit (effective hole size) increases as a function of time during the initial stages of gradual osmotic **hemolysis**. The process of gradual osmotic **hemolysis** is discussed in terms of mol. sieving through stress-induced effective membrane holes. It is suggested that a portion of the membrane protein might form an elastic network which would account for the gradual increase in size and apparent homogeneity of the effective holes.

L6 ANSWER 13 OF 13 CA COPYRIGHT 1997 ACS
AN 78:82705 CA
TI Serum myokinase-(adenylate kinase) activity in intravascular **hemolysis**
AU Mainzer, Klaus; Morsches, Bernhard; Holzmann, Hans
CS II. Med. Universitaetsklin. Poliklin., Mainz, Ger.
SO Aerztl. Forsch. (1972), 26(12), 426-31
CODEN: ARZFAN
DT Journal
LA German
AB The myokinase (MK) activity in the serum averaged 10.3 units/l. for humans and 15.1 for rabbits. In the **erythrocytes** it was 211.4 units/l. for humans and only 59.8 for rabbits. No creatine phosphokinase (CPK) was found in the **erythrocytes**. When a human **erythrocyte** suspension was treated with 0-0.7% NaCl, free Hb was found even at the lowest NaCl level; on the whole, the Hb and MK curves ran parallel. When rabbits were treated with phenylhydrazine for 4 days to induce **hemolysis**, the MK activity in the serum rose sharply as the Hb dropped, with a peak on the 5th day and a return to normal on the 10th day. The reticulocytes and the av. cell vol. increased, with a peak on the 8th day. Two patients with severe intravascular **hemolysis**

(due to ingestion of HOAc and water aspiration after drowning) also showed sharp increases of MK activity (to 100 and 25.4 units/l.), but values returned to normal the next day. In contrast to the in vitro results, the free Hb values remained higher after the MK had already dropped. The detn. of MK and free Hb together in the blood permits differentiation between muscular disease and **hemolysis**. The free Hb is a very sensitive indicator for intravascular **hemolysis** and the MK detn. will diagnose muscular diseases.

```
=> s l4(10a)(determination or quantitation)/ab,bi
          437 DETERMINATION/AB
       345825 DETERMINATION/BI
        15499 QUANTITATION/AB
        20927 QUANTITATION/BI
L7          1 L4(10A)(DETERMINATION OR QUANTITATION)/AB,BI

=> d bib,ab

L7   ANSWER 1 OF 1  CA  COPYRIGHT 1997 ACS
AN   115:275114  CA
TI   Quantitative organization of the known protein x-ray structures.  I.
     Methods and short-length-scale results
AU   Rackovsky, S.
CS   Sch. Med. Dent., Univ. Rochester, Rochester, NY, 14642, USA
SO   Proteins:  Struct., Funct., Genet. (1990), 7(4), 378-402
     CODEN: PSFGEY; ISSN: 0887-3585
DT   Journal
LA   English
AB   The problem of delineating the relationships between the known
     protein structures was addressed.  In order to study this problem,
     methods have been developed to represent arbitrarily sized fragments
     of biopolymer backbone, and to compare distributions of such
     fragments.  These methods are applied to a classification of 123
     structures representing the entire set of known x-ray structures.
     The resulting data are analyzed (on the four-C.alpha. length scale)
     to det. both the large-scale organization of the set of known
     structures (i.e., the relationships between large groups of
     structures, each comprised of proteins that are structurally
     related) and its local structure (i.e., the quant. degree of
     similarity between any two specific structures).  It is shown that
     the set of structures forms a continuum of structural types, ranging
     from all-helical to all-sheet/barrel proteins.  It is further
     demonstrated that the d. of protein structures is not uniform across
     this continuum, but rather that structures cluster in certain
     regions, sepd. by regions of lower population.  The properties of
     the various regions of the structural space are detd.  The existence
     is demonstrated of strong quant. correlations between the contents
     of different types of four-C.alpha. fragments within protein
     structures, which imply significant constraints on the types of
     architecture that can occur in proteins.  Anal. of the distribution
     of structures demonstrates some hitherto unsuspected similarities
     and suggests that, in some circumstances, neither structural
     similarity nor sequence homol. may be necessary conditions for
     evolutionary relationship between proteins.  It is also suggested
     that these unsuspected similarities may imply similar folding
     mechanisms for structures of apparently different global
     architecture.  Cases are also noted in which apparently similar
     structures may fold by different mechanisms.  The connection between
     structure and dynamic properties is discussed, and a possible role
     of dynamics in the evolution of protein structures is suggested.
     The sensitivity of the methods presented herein to anomalies of
     structure refinement is demonstrated.  It is suggested that the
     present results provide a framework for analyzing exptl. results on
     structural similarity obtained using vibrational CD spectra, which
     are sensitive to local backbone structure.

=> file reg
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
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DICTIONARY FILE UPDATES: 15 MAY 97 HIGHEST RN 188963-46-4

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=> d l2

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 1997 ACS
RN 9013-02-9 REGISTRY
CN Kinase (phosphorylating), adenylate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5'-AMP kinase
CN **Adenylate kinase**
CN Adenylic kinase
CN Adenylokinase
CN AMP kinase
CN E.C. 2.7.4.3
CN Kinase (phosphorylating), myo-
CN Myokinase
DR 9026-71-5
MF Unspecified
CI MAN
LC STN Files: AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, CA, CABA, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CJACS, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, PROMT, TOXLIT, USPATFULL
Other Sources: EINECS**, TSCA**
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22 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1881 REFERENCES IN FILE CAPLUS (1967 TO DATE)

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-6.44

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TERMINAL (ENTER 1, 2, 3, 4, OR ?):3p3.00

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  * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * *
  *                Help Desk --> 703-305-9000                     *
  *                                                               *
  *    The Help Desk is staffed for APS support 7 days/week.      *
  *      Monday through Friday:      6:30am - 9:00pm              *
  *      Saturday, Sunday, Holidays: 8:30am - 5:00 pm             *
  *                                                               *
  *    The Help Desk staff at this number will handle all APS     *
  *    related questions.                                         *
  *                                                               *
  * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * *
  *                                                               *
  *    >>>>>>>>>>>>> NEW SUNDAY HOURS !!! <<<<<<<<<<<<<           *
  *                                                               *
  *    The APS is available:                                      *
  *            6:30am - 9:00pm Monday through Friday              *
  *            7:30am - 5:00pm Saturday, Sunday, Holidays         *
  *                                                               *
  *      APS is unavailable Thanksgiving Day, Christmas Day,      *
  *      and New Year's Day.                                      *
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FILE 'USPAT' ENTERED AT 11:43:13 ON 16 MAY 1997

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  *             W E L C O M E   T O   T H E                  *
  *        U. S.   P A T E N T   T E X T   F I L E           *
  * * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

=> s ( (adenylate(w)kinase) or (amp(w)kinase) or ( adenylic(w)kinase ) or  (adenylokinase) or
(myokinase) ) (p) (hemolysis)
           779 ADENYLATE
          5181 KINASE
         20723 AMP
          5181 KINASE
           171 ADENYLIC
          5181 KINASE
             0 ADENYLOKINASE
            73 MYOKINASE
          2136 HEMOLYSIS
L1           2 ( (ADENYLATE(W)KINASE) OR (AMP(W)KINASE) OR ( ADENYLIC(W)KI
NAS
               E ) OR  (ADENYLOKINASE) OR (MYOKINASE) ) (P) (HEMOLYSIS)

=> d cit,kwic 1-2

1.  5,543,330, Aug. 6, 1996, Method for diagnosing myofibrogranuloma
(MFG) in walleye; Harry L. Holloway, Jr., et al., 436/79; 119/216;
436/63, 98, 811 [IMAGE AVAILABLE]

US PAT NO:     5,543,330 [IMAGE AVAILABLE]            L1: 1 of 2

DETDESC:

DETD(57)

 Creatine . . . (2100 U/L) were used as controls for testing
procedures. Some assays did not proceed in a linear fashion due to
hemolysis, which caused high levels of adenylate kinase, ATP
and glucose-6-phosphate to be released from red cells affecting the
reaction. These assays were not considered in the analysis.. . .

2.  4,912,033, Mar. 27, 1990, Creatine kinase MB determination method;
Jack H. Ladenson, et al., 435/7.4, 172.2, 337; 436/548; 530/388.25,
388.26, 808, 809; 935/103, 110 [IMAGE AVAILABLE]

US PAT NO:     4,912,033 [IMAGE AVAILABLE]            L1: 2 of 2

DETDESC:

DETD(103)

 The . . . serum was present than when only buffer was present. The
addition of lysed red blood cells as a source of adenylate kinase
did not influence the results even when the hemoglobin (as an assessment
of hemolysis) was as high as 246 g/l. See FIG. 3. The addition of up
to 200,000 U/l of CK-MM or 250,000. . .

=> s ( (adenylate(w)kinase) or (amp(w)kinase) or ( adenylic(w)kinase ) or  (adenylokinase) or
(myokinase) ) (p) (erthrocyt? or red(2w)cell#)
           779 ADENYLATE
          5181 KINASE
         20723 AMP
          5181 KINASE
           171 ADENYLIC
          5181 KINASE
             0 ADENYLOKINASE
            73 MYOKINASE
```

```
          126 ERTHROCYT?
       129592 RED
       222274 CELL#
L2           3 ( (ADENYLATE(W)KINASE) OR (AMP(W)KINASE) OR ( ADENYLIC(W)KI
NAS
               E ) OR  (ADENYLOKINASE) OR (MYOKINASE) ) (P) (ERTHROCYT? OR
 RE
               D(2W)CELL#)

=> d 1-3 cit,kwic

1.  5,543,330, Aug. 6, 1996, Method for diagnosing myofibrogranuloma
(MFG) in walleye; Harry L. Holloway, Jr., et al., 436/79; 119/216;
436/63, 98, 811 [IMAGE AVAILABLE]

US PAT NO:     5,543,330 [IMAGE AVAILABLE]            L2: 1 of 3

DETDESC:

DETD(57)

 Creatine . . . for testing procedures. Some assays did not proceed in
a linear fashion due to hemolysis, which caused high levels of
adenylate kinase, ATP and glucose-6-phosphate to be released from
red cells affecting the reaction. These assays were not
considered in the analysis. A frequency distribution of the data was made
and. . .

2.  4,912,033, Mar. 27, 1990, Creatine kinase MB determination method;
Jack H. Ladenson, et al., 435/7.4, 172.2, 337; 436/548; 530/388.25,
388.26, 808, 809; 935/103, 110 [IMAGE AVAILABLE]

US PAT NO:     4,912,033 [IMAGE AVAILABLE]            L2: 2 of 3

DETDESC:

DETD(103)

 The . . . from the latex beads was greater when serum was present
than when only buffer was present. The addition of lysed red blood
cells as a source of adenylate kinase did not influence the
results even when the hemoglobin (as an assessment of hemolysis) was as
high as 246 g/l.. . .

3.  4,220,714, Sep. 2, 1980, Composition for inhibiting adenylate-kinase
and its use; Franco Meiattini, et al., 435/17, 26, 184 [IMAGE AVAILABLE]

US PAT NO:     4,220,714 [IMAGE AVAILABLE]            L2: 3 of 3

SUMMARY:

BSUM(31)

              TABLE 2

Effect of AMP 2 mmol/liter and Fluoride 6 mmol/liter
on Adenylate Kinase activity from different sources
                      plus AMP 2 mmol/l and
Source of
         Szasz method (10)
                      fluoride 6 mmol/l*.  .  .  6154       83
heart    4433         613         86
Human liver
         1133         58          95
muscle   7802         245         97
heart    1275         49          96
erthrocytes
         978          40          96
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platelets
285 7 98

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*The indicated concentration of AMP and the fluoride have. . .

=> s l1 and l2
L3 2 L1 AND L2

=> d

1. 5,543,330, Aug. 6, 1996, Method for diagnosing myofibrogranuloma (MFG) in walleye; Harry L. Holloway, Jr., et al., 436/79; 119/216; 436/63, 98, 811 [IMAGE AVAILABLE]

=> d 2

2. 4,912,033, Mar. 27, 1990, Creatine kinase MB determination method; Jack H. Ladenson, et al., 435/7.4, 172.2, 337; 436/548; 530/388.25, 388.26, 808, 809; 935/103, 110 [IMAGE AVAILABLE]

=> logoff y

U.S. Patent & Trademark Office LOGOFF AT 11:46:20 ON 16 MAY 1997

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ATHZ
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